Set Name side by side	Query	Hit Count	Set Name result set
DB=JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L23	(cd30L or cd30 adj ligand) and (tumor$ or tumour$ or cancer$)	4	L23
L22	(cd30L or cd30 adj ligand) and (tumor$ or tumour$ or cancer$) and photodynamic	0	L22
L21	(cd40L or cd40 adj ligand or gp39) and (tumor$ or tumour$ or cancer$) and photodynamic	1	L21
L20	L19 and photodynamic	1	L20
L19	(cd40L or cd40 adj ligand or gp39) same (tumor$ or tumour$ or cancer$)	32	L19
L18	(cd30L or cd30 adj ligand) same (tumor$ or tumour$ or cancer$)	2	L18
DB=USPT; PLUR=YES; OP=ADJ			
L17	(cd40L or cd40 adj ligand or gp39) same (tumor$ or tumour$ or cancer$) same (treat$ or therap$ or administ$)	70	L17
DB=USPT,PGPB; PLUR=YES; OP=ADJ			
L16	(cd40L or cd40 adj ligand or gp39) same (tumor$ or tumour$ or cancer$) same (treat$ or therap$ or administ$)	113	L16
L15	L12.clm.	3	L15
L14	L12 and (cd30L ro cd30 adj ligand)	0	L14
L13	L12 and photodynamic	8	L13
L12	(cd40L or cd40 adj ligand or gp39) same (tumor$ or tumour$ or cancer$) same (treat$ or inhibit$ or suppress$ or therap$ or administ$)	193	L12
L11	(cd30L or cd30 adj ligand) same (tumor$ or tumour$ or cancer$) and photodynamic	2	L11
L10	L9 and (cd40L or cd40 adj ligand or cd40)	11	L10
L9	L8 same (treat$ or therap$ or prevent$ or administ$)	16	L9
L8	(cd30L or cd30 adj ligand) same (tumor$ or tumour$ or cancer$)	87	L8
L7	(cd30L or cd30 adj ligand) and (tumor$ or tumour$ or cancer$)	259	L7
L6	L2 and (tumor$ or tumour$ or cancer$)	1	L6
L5	L1 and (tumor$ or tumour$ or cancer$)	14	L5
L4	L3 and (tumor$ or tumour$ or cancer$)	12	L4
L3	L1 and (cd40L or cd40 adj ligand)	12	L3
L2	L1 and (cd30L or cd30 adj ligand)	1	L2
L1	fanslow-william$	21	L1

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Term	Documents
CD30L.DWPI,EPAB,JPAB.	2
CD30LS	0
CD30.DWPI,EPAB,JPAB.	49
CD30S	0
LIGAND.DWPI,EPAB,JPAB.	23176
LIGANDS.DWPI,EPAB,JPAB.	9989
TUMOR$	0
TUMOR.DWPI,EPAB,JPAB.	14866
TUMORACTIVITY.DWPI,EPAB,JPAB.	1
TUMORAL.DWPI,EPAB,JPAB.	199
TUMORALLY.DWPI,EPAB,JPAB.	3
((CD30L OR CD30 ADJ LIGAND) AND (TUMOR$ OR TUMOUR$ OR CANCER$)).JPAB,EPAB,DWPI.	4

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DATE: Tuesday, December 31, 2002 Printable Copy Create Case

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L10: Entry 1 of 11 File: PGPB Dec 19, 2002

DOCUMENT-IDENTIFIER: US 20020192222 A1
TITLE: Receptor specific transepithelial transport of therapeutics

Detail Description Paragraph (85):
[0111] Other supplementary immune potentiating agents, such as cytokines, may be delivered in conjunction with the conjugates of the invention. The cytokines contemplated are those that will enhance the beneficial effects that result from administering the immunomodulators according to the invention. Cytokines are factors that support the growth and maturation of cells, including lymphocytes. It is believed that the addition of cytokines will augment cytokine activity stimulated in vivo by carrying out the methods of the invention. The preferred cytokines are interleukin (IL)-1, IL-2, gamma-interferon and tumor necrosis factor .alpha.. Other useful cytokines are believed to be IL-3, IL-4, IL-5, IL-6, IL-7, IL-8, IL-9, IL-10, IL-11, IL-12, IL-13, erythropoietin, leukemia inhibitory factor, oncostatin-M, ciliary neurotrophic factor, growth hormone, prolactin, CD40-ligand, CD27-ligand, CD30-ligand, alpha-interferon, beta-interferon, and tumor necrosis factor-.beta.. Other cytokines known to modulate T-cell activity in a manner likely to be useful according to the invention are colony stimulating factors and growth factors including granulocyte and/or macrophage stimulating factors (GM-CSF, G-CSF and CSF-1) and platelet derived, epidermal, insulin-like, transforming and fibroblast growth factors. The selection of the particular cytokines will depend upon the particular modulation of the immune system that is desired. The activity of cytokines on particular cell types is known to those of ordinary skill in the art.

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L10: Entry 4 of 11 File: PGPB May 30, 2002

PGPUB-DOCUMENT-NUMBER: 20020065210
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020065210 A1

TITLE: DcR3 polypeptide, a TNFR homolog

PUBLICATION-DATE: May 30, 2002

US-CL-CURRENT: 514/1; 424/143.1, 435/320.1, 435/325, 435/69.1, 530/350, 536/23.5, 800/14, 800/18

APPL-NO: 09/ 894924 [PALM]
DATE FILED: June 28, 2001

RELATED-US-APPL-DATA:
Application 09/894924 is a continuation-of US application 09/157289, filed September 18, 1998, ABANDONED
Application is a non-provisional-of-provisional application 60/059288, filed September 18, 1997,
Application is a non-provisional-of-provisional application 60/094640, filed July 30, 1998,

RELATED APPLICATIONS

[0001] This application is a non-provisional application claiming priority under Section 119(e) to provisional application number 60/059,288 filed Sep. 18, 1997 and to provisional application number 60/094,640 filed Jul. 30, 1998, the contents of which are hereby incorporated by reference.

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L10: Entry 4 of 11 File: PGPB May 30, 2002

DOCUMENT-IDENTIFIER: US 20020065210 A1
TITLE: DcR3 polypeptide, a TNFR homolog

Summary of Invention Paragraph (4):
[0003] Various molecules, such as tumor necrosis factor-.alpha. ("TNF-.alpha."), tumor necrosis factor-.beta. ("TNF-.beta." or "lymphotoxin"), CD30 ligand, CD27 ligand, CD40 ligand, OX-40 ligand, 4-1BB ligand, Fas ligand (also referred to as Apo-1 ligand or CD95 ligand), and Apo-2 ligand (also referred to as TRAIL) have been identified as members of the tumor necrosis factor ("TNF") family of cytokines [See, e.g., Gruss and Dower, Blood, 85:3378-3404 (1995); Wiley et al., Immunity, 3:673-682 (1995); Pitti et al., J. Biol. Chem., 271:12687-12690 (1996)]. Among these molecules, TNF-.alpha., TNF-.beta., CD30 ligand, 4-1BB ligand, Fas ligand, and Apo-2 ligand (TRAIL) have been reported to be involved in apoptotic cell death. Both TNF-.alpha. and TNF-.beta. have been reported to induce apoptotic death in susceptible tumor cells [Schmid et al., Proc. Natl. Acad. Sci., 83:1881 (1986); Dealtry et al., Eur. J. Immunol., 17:689 (1987)]. Zheng et al. have reported that TNF-.alpha. is involved in post-stimulation apoptosis of CD8-positive T cells [Zheng et al., Nature, 377:348-351 (1995)]. Other investigators have reported that CD30 ligand may be involved in deletion of self-reactive T cells in the thymus [Amakawa et al., Cold Spring Harbor Laboratory Symposium on Programmed Cell Death, Abstr. No. 10, (1995)].

Summary of Invention Paragraph (9):
[0008] A similar repetitive pattern of CRDs exists in several other cell-surface proteins, including the p75 nerve growth factor receptor (NGFR) [Johnson et al., Cell, 47:545 (1986); Radeke et al., Nature, 325:593 (1987)], the B cell antigen CD40 [Stamenkovic et al., EMBO J., 8:1403 (1989)], the T cell antigen OX40 [Mallet et al., EMBO J., 9:1063 (1990)] and the Fas antigen [Yonehara et al., supra and Itoh et al., Cell, 66:233-243 (1991)]. CRDs are also found in the soluble TNFR (sTNFR)-like T2 proteins of the Shope and myxoma poxviruses [Upton et al., Virology, 160:20-29 (1987); Smith et al., Biochem. Biophys. Res. Commun., 176:335 (1991); Upton et al., Virology, 184:370 (1991)]. Optimal alignment of these sequences indicates that the positions of the cysteine residues are well conserved. These receptors are sometimes collectively referred to as members of the TNF/NGF receptor superfamily. Recent studies on p75NGFR showed that the deletion of CRD1 [Welcher, A. A. et al., Proc. Natl. Acad. Sci. USA, 88:159-163 (1991)] or a 5-amino acid insertion in this domain [Yan, H. and Chao, M. V., J. Biol. Chem., 266:12099-12104 (1991)] had little or no effect on NGF binding [Yan, H. and Chao, M. V., supra]. p75 NGFR contains a proline-rich stretch of about 60 amino acids, between its CRD4 and transmembrane region, which is not involved in NGF binding [Peetre, C. et al., Eur. J. Hematol., 41:414-419 (1988); Seckinger, P. et al., J. Biol. Chem., 264:11966-11973 (1989); Yan, H. and Chao, M. V., supra]. A similar proline-rich region is found in TNFR2 but not in TNFR1.

Summary of Invention Paragraph (11):
[0010] The TNF family ligands identified to date, with the exception of lymphotoxin-.alpha., are type II transmembrane proteins, whose C-terminus is extracellular. In contrast, most receptors in the TNF receptor (TNFR) family identified to date are type I transmembrane proteins. In both the TNF ligand and receptor families, however, homology identified between family members has been found mainly in the extracellular domain ("ECD"). Several of the TNF family cytokines, including TNF-.alpha., Fas ligand and CD40 ligand, are cleaved proteolytically at the cell surface; the resulting protein in each case typically

forms a homotrimeric molecule that functions as a soluble cytokine. TNF receptor family proteins are also usually cleaved proteolytically to release soluble receptor ECDs that can function as inhibitors of the cognate cytokines.

Detail Description Paragraph (120):
[0162] In methods of treating cancer using the DcR3 antagonists described herein, it is contemplated that other, additional therapies may be administered to the mammal, and such includes but is not limited to, chemotherapy and radiation therapy, immunoadjuvants, cytokines, and antibody-based therapies. Examples include interleukins (e.g., IL-1, IL-2, IL-3, IL-6), leukemia inhibitory factor, interferons, TGF-beta, erythropoietin, thrombopoietin, HER-2 antibody and anti-CD20 antibody. Other agents known to induce apoptosis in mammalian cells may also employed, and such agents include TNF-.alpha., TNF-.beta. (lymphotoxin-.alpha.), CD30 ligand, and 4-1BB ligand.

L8: Entry 66 of 87 File: USPT Sep 10, 2002

DOCUMENT-IDENTIFIER: US 6448390 B1
TITLE: Stable envelope proteins for retroviral, viral and liposome vectors and use in gene drug therapy

Detailed Description Text (255):
In another example, we will enable the use of two motifs in stabilizing a retroviral vector for targeted gene delivery to human Hodgkin's lymphoma cells and other human malignancies of hematopoietic origin. Hodgkin's lymphoma cells and cells of other human hematopoietic malignancies express high levels of CD30, the receptor for human CD30Ligand (CD30L) (Durkop et. al., 1992 Cell 68: 421-427; Smith et. al., 1993 Cell 73: 1349-1360; and Gattei et. al., 1997 Blood 89: 2048-2059). Thus, CD30 provides a particularly attractive receptor for virus- or liposome-mediated human gene and drug therapy of a wide range of hematopoietic malignancies using the CD30L as a targeting sequence because cancer cell killing will result from retroviral vector binding, as the interaction of CD30L with CD30 initiates a signal transduction cascade that causes programmed cell death, as well as from delivery of a lethal gene or drug. The recombinant retroviral vector genomes for this example are constructed by inserting the nucleotide sequence encoding the 360 amino acid receptor-binding ectodomain of human CD30L in-frame into the unique Not I site of the four basic retroviral vectors described above using standard PCR techniques. Virus binding is quantitated on mouse NIH3T3, African green monkey COS-1, human 293, human cutaneous T cell lymphoma HUT-102 (ATCC TIB-162) from which a CD30 cDNA is isolated, and human 293 cells expressing the CD30 cDNA. In this example, specific binding of virus to the cognate receptor (CD30) of the fused targeting sequence (CD30L) is quantitated by measuring the decrease in mean fluorescence observed when virus and cells are incubated in the presence of antibody specific for the fused CD30L targeting sequence as well as for the CD30L binding site on CD30, its cognate receptor. The preferred vector is expected to bind specifically to HUT-102 and 293 cells expressing high levels of CD30 and lacking the ecotropic MLV receptor, but not to 293 and COS-1 cells which lack CD30 and the ecotropic MLV receptor, regardless of binding to NIH3T3 cells. Titration of all virus stocks are performed on the same cell lines. The preferred vector is expected to deliver the .beta.-galactosidase gene to the greatest number of HUT-102 and 293 cells expressing high levels of CD30, with the lowest ratio of gene delivery to 293 and COS-1 cells compared to delivery to the CD30-expressing cells. The preferred vector to be used for gene delivery in vivo is expected to deliver the .beta.-galactosidase gene to the greatest number of HUT-102 cells upon inspection of the bone marrow of SCID mice injected with HUT-102 prior to retroviral vector administration, with the lowest ratio of gene delivery to mouse tissues.

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L8: Entry 72 of 87 File: USPT Nov 7, 2000

DOCUMENT-IDENTIFIER: US 6143869 A
TITLE: CD30 ligand oligomers and polypeptides

Brief Summary Text (64):
Preferred therapeutic agents are radionuclides and drugs. In one embodiment of the invention, the anti-tumor drug calicheamycin is attached to a soluble human CD30 ligand polypeptide.

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L17: Entry 6 of 70 File: USPT Aug 6, 2002

DOCUMENT-IDENTIFIER: US 6429199 B1
TITLE: Immunostimulatory nucleic acid molecules for activating dendritic cells

Detailed Description Text (81):
It is also contemplated according to the methods of the invention that any compositions of the invention may also be administered in conjunction with other immune stimulating agents, such as for instance cytokines. Cytokines, include but are not limited to, IL-1, IL-2, IL-4, IL-5, IL-6, IL-7, IL-10, IL-12, IL-1 5, granulocyte-macrophage colony stimulating factor (G-MCSF), granulocyte colony stimulating factor (GCSF), interferon-y (IFN-.gamma.), tumor necrosis factor (TNF), TGF-.beta., FLT-3 ligand, and CD40 ligand.

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L17: Entry 8 of 70 File: USPT Jul 23, 2002

DOCUMENT-IDENTIFIER: US 6423539 B1
TITLE: Adjuvant treatment by in vivo activation of dendritic cells

Brief Summary Text (12):
Methods are provided for enhancing the immunogenicity of an antigen by increasing the specific antigen presenting function of DC in a mammalian host. Prior to the immunization with an antigen, the host is treated with a DC mobilization agent, e.g. Flt-3 ligand, GM-CSF, G-CSF/FIt3L fusion protein, etc. This treatment effectively increases the number of circulating DC precursors. The host is then given a local, e.g. sub-cutaneous, intramuscular, etc., injection of antigen in combination with a DC activating agent, e.g. immunostimulatory DNA sequences, IL-1, alpha interferon, LPS, endotoxin, CD40L, poly IC, etc. The activation step promotes recruitment and maturation of the DC, along with antigen-specific activation and migration from the tissues to lymphoid organs. These DC then effectively interact with, and present processed antigen to, T cells that are then able to respond to the antigen. The methods of the invention are particularly useful in situations where the host response to the antigen is sub-optimal, for example in conditions of chronic infection, a lack of immune response to tumor antigens, and the like. In one aspect of the invention, the antigen is a tumor antigen, and is used to enhance the host immune response to tumor cells present in the body.

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L17: Entry 13 of 70 File: USPT Apr 23, 2002

DOCUMENT-IDENTIFIER: US 6376654 B1
TITLE: Myeloma cell and ovarian cancer cell surface glycoproteins, antibodies thereto, and uses thereof

Detailed Description Text (19):
In the area of multiple myeloma, the antibodies or fragments of the present invention have further utility in the preparation of cellular samples from which myeloma cells have been removed. This is particularly important in autologous bone marrow transplants, wherein a sample of bone marrow is harvested from a cancer patient prior to the patient undergoing high-dose chemotherapy. The goal of the high dose chemotherapy is to destroy the cancer cells, which also results in the depletion of bone marrow cells. Following such treatment the harvested bone marrow cells are reintroduced to the patient. In myeloma and related diseases, the harvested bone marrow is contaminated with myeloma cells; reintroduction of untreated bone marrow will simply reintroduce the disease. Previous methods to prevent reintroduction of cancer cells have included treatment of the bone marrow sample with chemotherapeutic agents and other anti-neoplastic agents in vitro; other methods include purging the sample of cancer cells. In a further practice of the present invention, the monoclonal antibodies and fragments described herein may be used to remove myeloma cells from a bone marrow sample before reintroduction to the patient. In one non-limiting example, the monoclonal antibody or binding fragments are attached to a matrix such as beads. This may be accomplished by any of several well-known methods for preparing an affinity matrix comprising antibodies or binding fragments. The bone marrow sample is exposed to the matrix, such as by passage of the cells over a column containing the matrix, under conditions to promote the binding of the myeloma cells in the sample through antigen/antibody interactions with the antibody or binding fragments on the matrix. The myeloma cells in the sample adhere to the matrix; the column effluent or non-adherent cellular population is depleted of myeloma cells. The effectiveness of the procedure may be monitored by examining the cells for residual myeloma cells, such as by using a detectably-labeled antibody as described below. The procedure may be repeated or modified to increase effectiveness. This purging procedure [see, e.g., Ramsay et al., J. Clin. Immunol., 8(2): 81-88 (1988)] may be performed together with other methods for removing or killing cancer cells, including but not limited to exposing the purified bone marrow cells to chemotherapeutic agents. Such chemotherapeutic agents include the use of the antibodies or binding fragments of the present invention conjugated to a cytotoxic agent, as described above for in-vivo therapeutic treatment. Thus, conjugates of the antibodies or fragments of the invention with cytotoxic agents may be used for the ex-vivo killing of tumor cells in a cellular sample. These methods may include additionally exposing the cells to cytokines (GM-CSF, IL-6), cytokine receptors (such ad IL-6-receptor), mitogens (such as PWM-Poke weed mitogen) or adhesion molecules (such as CD40 ligand) in order to stimulate the myeloma cells to rapidly differentiate and thereby upregulate expression of cancer-specific antigens on their cell surface. These treatment modalities are aimed to render the myeloma cells vulnerable to the in-vitro mediated cytotoxicity instigated by incubation with the monoclonal antibody.

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L17: Entry 23 of 70 File: USPT May 15, 2001

DOCUMENT-IDENTIFIER: US 6232088 B1
TITLE: Treatment and prevention of immune rejection reactions

Brief Summary Text (8):
The invention further provides (a) methods for treating or prophylactically preventing a cell-cell or cell-virus adhesion syndrome comprising administering an anti-adhesion effective amount of a hydrolase effective to remove or inactivate a cellular or viral acceptor or receptor adhesion component that is involved in the cell-cell or cell-virus adhesion, (b) compositions or substances for use in such methods, (c) pharmaceutical compositions containing effective amounts of enzyme for use in such methods, and (d) uses of the enzyme composition for manufacturing a medicament for use in such methods. Preferably, the syndrome comprises inflammation, shock, tumor metastases, autoimmune disease, transplantation rejection reactions or microbial infections. Preferably, (a) the syndrome is selected from the group consisting of graft versus host disease, organ or tissue transplantation rejection, autoimmune disease and associated conditions, microbial infection, immune disorder, cystic fibrosis, COPD, atherosclerosis, cancer, asthma, septic shock, toxic shock syndrome, conjunctivitis, reperfusion injury and pain, and (b) a cell surface receptor, associated with the cell-cell or cell-virus adhesion syndrome, is removed or inactivated by the administered hydrolase, where the cell surface receptor can be selected from the group consisting of ICAM-1, ICAM-2, VCAM-1, CD3, CD4, CD8, CD11, CD18, CD28, CD29D, CD31, CD44, CD49, CD62L, CD102, GP39, integrins and asialo GM1 ceramide.